hypodermic injection of morphin gives additional comfort. The patient should be kept absolutely quiet in bed on restricted diet, until the decline of the fever. Internal medication is indicated by special symptoms. Diaphoretics are sometimes useful for modifying the pyrexia in the early stage of the disease. After the decline of the fever, the effusion having apparently reached its maximum, and when scanty secretion of the kidneys is still an existing symptom, the exhibition of diuretics is called for to stimulate absorption of the fluid. The bowels should also be kept freely evacuated, administering, if required, saline laxatives occasionally preceded by calomel followed by a purgative. The operation of thoracentesis should be performed in all cases in which the pleural cavity is largely filled with fluid, and in others with excessive dyspnea or when signs of absorption fail to appear. The internal and external use of iodin and finally of muriated tincture of iron will hasten absorption of fluid and inflammatory thickening of the pleuræ.

Pus in the pleural cavity should be promptly removed by free incision and rib resection to establish free drainage. An unexpanded lung and consequent retraction of one side of the chest are subject to improvement or cure by pulmonary gymnastics and systemitized exercise of the body in general.

RECURRENT PNEUMOTHORAX: REPORT OF A CASE, WITH REVIEW OF THE LITERATURE.

By Clyde L. Cummer, M.D.,
CLEVELAND, OHIO.

Medical literature is replete with studies of pneumothorax, and although cases of presumed spontaneous pneumothorax are not specially common, the subject has been well considered. In attempting to ascertain the frequency of recurrent pneumothorax we found that little had been written concerning it. In Albutt's *System,*[1] Finlay says that "as a curiosity attention may be directed to recurring pneumothorax: these cases are accompanied by few symptoms, nearly always recover, and may therefore be due to rupture of emphysematous bullæ." Neither in Osler's *Modern Medicine*[2] nor in Nothnagel's *Encyclopedia of Practical Medicine*[3] is even mention made of this phase of the subject under the general head of "pneumothorax."

In 1888 Gabb[4] reported a case which he had seen in one attack of pneumothorax. The patient, a female, had had three preceding attacks and had been under the care of Dr. Byrom Bramwell and his father. Her age at the time of the first attack, fourteen years previous, was forty-two. She had also had bronchitis and pleurisy of the right side in addition to the attacks of pneumothorax. In this case the pneumothorax had appeared on the same side each time. In the first attack air filled the entire right pleural cavity, but in the second the pneumothorax was localized and limited. Dr. Bramwell had been unable to find any evidence of tuberculosis, but stated that the family history was bad. One sister had a well-marked phthisis at the time of the patient's first pneumothorax. Byrom Bramwell advanced the theory that the first attack was due to the pulling upon an adhesion at the right apex during violent effort, so rupturing the lung. As the air was absorbed a friction rub appeared. Three of the attacks were on the same side. The location of one is not stated.

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[1] Albutt and Rolleston, 1909, v, 575.

[2] Walter R. James, iii, 868.

[3] O. Rosenbach, Pneumothorax, p. 972.

[4] Recurrent Pneumothorax, British Med. Jour., 1888, ii, 178.

In 1892 Vitvitski[5] saw a young man, tailor by occupation, who complained of shortness of breath, cough, and general weakness. The family history was unimportant, except that his father died at the age of forty years of tuberculosis. A year preceding this illness the patient had noted cough and pain in the chest. His pains were temporary at first, but his health soon became so much impaired that he had to give up his occupation. At one time there appeared very suddenly dyspnea, with considerable pain in the left chest. This shortness of breath, however, passed in a few days. Two months previous to his admission to the hospital he began to have diarrhea and vomiting after taking food. Physical examination showed that he had a left-sided pneumothorax with cavity formation on the right side, well-marked signs of general pulmonary tuberculosis, and tuberculosis of the kidney. He died eight days after admission to the hospital, and the clinical findings were confirmed by the autopsy. Examination of the left chest showed not only a pneumothorax but a scar in the pleura, the size of a lentil evidently the location of the perforation of the preceding pneumothorax.

In 1898 Finny[6] recounted the history of a young man whom he had observed in two different attacks. His age at the time of his first admission to the hospital in 1897 was eighteen. The pneumothorax was on the left side. He was discharged from the hospital about five weeks later. During the absorption of air, dry pleurisy appeared. Two weeks after his discharge he reappeared at the hospital, with return of the signs and symptoms on the same side. There is no record of subsequent observation. It is of interest to note that no family history of tuberculosis was obtained.

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[5] A Case of Recurring Unilateral Pneumothorax, Vrach, St. Petersburg, 1894, xv, 960.

[6] Dublin Jour. Med. Sci., 1898, cv, 273.

In 1907 Sale[7] reported a remarkable case which had come under his observation. The patient, a young woman, aged twenty-two years, had had eleven attacks of pneumothorax. In her case there was no history of familial tuberculosis. The first attack was preceded by a period of coughing and spitting of blood. There had been no fluid in the chest at any time. He did not consider that a tuberculosis infection was the causative factor.

In 1908 Hamilton[8] reported the subsequent course of 4 cases of pneumothorax whose histories had been given in a previous article. One of these cases had two attacks of pneumothorax, both on the right side. Ten years after the last attack the patient was in good health and had had no recurrence of the condition. There was nothing in this case which Hamilton considered indicative of a tubercle infection. He was under observation eleven years.

The following case came under my observation: J. H., unmarried male, aged twenty-three years. To my personal knowledge he had been in excellent health since childhood. In the family history the only significant point was the fact that his only brother had suffered at one time, some years previously, from a tuberculous elbow. This had healed after immobilization. His father and only sister were in good health. His mother died of cardiac disease. He walked to my residence on February 13, 1912, complaining of severe pain in the right side. It appeared suddenly the same day while taking his morning bath. He had been in the enjoyment of his usual good health. Temperature 98° F., pulse 80, respiration 20. There was no evident dyspnea. Physical examination at once showed that the left side of the chest was distinctly more voluminous than the right. The supraclavicular space and the intercostal spaces on this side were filled out and the intercostal depressions were not visible. Respiratory movement was slight on both sides. On the affected side, tactile fremitus was absent, while the breath sounds were distant, high-pitched, of metallic quality, with inspiration and expiration of about equal duration and intensity. The heart was pushed over to the right, the right border of dulness being 5 cm. to the right of the midsternal line and the left border being 8 cm. to the left of the same line in the fifth interspace. The point of maximum cardiac impulse was in the sixth interspace, 4 cm. to the left of the midsternal line.

Aside from the pain in the chest the patient felt perfectly well and wanted to be out. He did not seem to be ill. He was sent home and kept at rest, but would not remain in bed. February 15 his general condition remained excellent. The white blood cells were 12,000 per c.mm. Examination of the chest showed that the left side was full and immobile. The lower border of the resonance was in the ninth interspace in the axillary line and in the eleventh interspace in the scapular line. I was unable to demonstrate the coin sound.

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[7] Lancet, 1907, ii, 1572.

[8] Spontaneous Pneumothorax, Montreal Med. Jour., 1908, xxxvii, 492.

On February 18 the patient developed a complicating follicular tonsillitis, in consequence of which the temperature rose to 101.4°. The chest showed decided change. Pulmonary resonance was demonstrated at the usual points. The heart had resumed practically its normal position. The breath sounds were fairly well heard over the entire left chest except at the base posteriorly. A distinct friction rub was heard in the lower portion of the axilla and below the heart when the patient was in the reclining posture. On February 21 (eight days after onset) the physical signs had largely disappeared. The friction rub in the axilla was less and the breath sounds were not so distinct. The patient resumed his usual duties as a surveyor in about three weeks after the onset of the pneumothorax. Frequent examinations failed to show any deviation from normal.

July 13, 1913, the patient consulted me again, complaining as before of pain in the chest. This had come on suddenly. The left chest looked much fuller than the right, but it moved well on respiration, and this time the heart was not displaced. Over the entire left chest the percussion note was slightly tympanitic except at the left base posteriorly, where it was dull. Over the entire left chest the breath sounds were very distant, except over the base posteriorly, where they were absent. Tacticle fremitus was lessened throughout; over the base it was absent. No adventitious sounds were heard.

July 19 the patient reported that the pain was less. The physical findings, however, were about the same.

July 24, Drs. W. C. Hill and G. F. Thomas made stereoscopic Roentgen-ray plates. They showed a pneumothorax on the left side with what was evidently a band of adhesions running between the right pleura and the pericardium. Shadows seen in the right chest were considered by Drs. Hill and Thomas to be caused by foci of healed tuberculosis. The patient was then reëxamined very carefully. On percussion the note over the apex of the right lung proved to be slightly shorter in duration and higher in pitch than normal. An expansion of 1.5 cm. was present at the apex. The breath sounds were normal except for slightly high pitch. Vocal and tacticle fremitus were normal throughout the right lung. No rales were heard anywhere.

August 7, the twenty-fifth day after onset, the patient reported that he suffered practically no pain. Physical examination showed that the signs of pneumothorax had practically cleared up. Resonance was the same on both sides and breath sounds were transmitted as well on one side as the other, with the exception that over the right apex posteriorly, resonance was somewhat deadened. On expansion the right apex did not move as freely as the left. Since this time the patient has been examined frequently.

July 23, 1914, no deviation from normal could be detected anywhere. His weight is and has been for some months at his normal maximum. He feels perfectly well, looks well, and is engaged daily in his occupation.

August 18, 1914, Drs. Hill and Thomas again made a Roentgen-ray picture, which showed normal relations between pleura and lungs. The amount of presumed tuberculous involvement was the same as in the previous finding.

In discussing this history my own conclusion is that a dormant tubercle infection explains the etiology. In favor of this view we have Roentgen-ray plates demonstrating the existence of apparently healed foci and the presence of slight though definite physical signs at one apex, together with a transitory, dry pleurisy. The band of adhesion running between the right pleura and the pericardium, as reported by the roentgenologist, is extremely interesting in connection with the theory advanced by Byrom Bramwell in one case (Gabb).[9] One is forced to admit that the continued good health of the patient weighs heavily against this theory. It is quite possible, however, that the pneumothorax has served in this case, and perhaps in all the other reported cases but one, as a therapeutic measure, and that its occurrence has tended to retard the flaring up of a slight tuberculous focus.

In analyzing the reports, which comprise all that I have been able to discover in the literature, we see that the accompanying history gives a total of six cases. Of this number four had only two attacks (Vitvitski,[10]; Finny,[11] Hamilton[12]), but of the three one was observed but a short time and one died. One case (Gabb)[13] had four attacks and one had eleven. In some instances only a few months elapsed between the disappearance of air in the pleural sac and its reappearance, while occasionally one or two years intervened.

It is of interest to note that in one case the time elapsing between the first attack and the fourth was fourteen years, and that Sale's[14] case had all of her eleven attacks in six years.

We can find no positive evidence that more than one side was affected in any instance. It is stated by Gabb[15] that the right side was the seat of the trouble three times. The location of one seizure is not noted. Finny,[16] Vitvitski,[17] and Hamilton[18] specify that the trouble made its second appearance on the same side. Sale[19] states that three attacks were on the right side, but fails to note the location of eight. The fact must have some significance in considering the pathology, and does not bear out the theory advanced by certain writers that so-called idiopathic pneumo-

[9] Loc. cit.

[10] Loc. cit.

[11] Loc. cit.

[12] Loc. cit.

[13] Loc. cit.

[14] Loc. cit.

[15] Loc. cit.

[16] Loc. cit.

[17] Loc. cit.

[18] Loc. cit.

[19] Loc. cit.

thorax is due to the rupture of emphysematous vesicles. If this were true the condition would be expected to affect the two sides impartially.

One case only, the one which terminated fatally, presented undeniable signs of infection with tubercle. Another had a family history of tuberculosis, and had had bronchitis and pleurisy in the interval between two manifestations of pneumothorax. A third case gave a history of cough and bloody expectoration preceding her first pneumothorax. One case showed Roentgen-ray findings pointing to tubercle infection. In the remaining two cases there was no apparent valid reason for suspecting the presence of a latent tuberculosis.

Conclusions regarding the etiology of recurrent pneumothorax would be out of place with so little data at hand. It is reasonable to suppose that it is the same as in cases of "spontaneous" pneumothorax. The etiology of this condition has been quite thoroughly discussed, West[20] making the point that tuberculosis is always the causative factor. This series of cases, with four showing definite or suggestive signs of tubercle infection, is not without interest.

A CLASSIFICATION AND ANALYSIS OF CLINICAL TYPES OF SPLENOMEGALY ACCOMPANIED BY ANEMIA.[1]

By E. B. Krumbhaar, M.D.,
OF PHILADELPHIA.

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania, Philadelphia, Pa.)

Chronic splenomegaly, usually associated with anemia, is not an uncommon condition both in adults and children. The forms without leukocytosis, more or less related to "splenic anemia," it is my intention to consider here, omitting the types of enlarged spleen in which other features render differentiation easy (such as leukemia, pseudoleukemia, polycythemia, and those secondary to obstruction or infection, as in heart disease, typhoid, kala-azar, syphilis, and so forth).

Splenic Anemia. The connection between chronic enlargement of the spleen and marked anemia without leukocytosis, as denoted in the term "splenic anemia," was first established forty-eight years ago by Gretzel,[2] in Griesinger's clinic, in Berlin. He described the

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[20] Samuel West, Pneumothorax, Lancet, 1897, i, 1264.

[1] Being part of an address read before the Joint Meeting of the Pediatric Societies, New York Academy of Medicine, November 5, 1914.

[2] Ein Fall von Anemia Splenica bei Einem Kind, Berl. klin. Woch., 1866, ii, 212.